# SOMATOSENSORY FUNCTION IN INDIVIDUALS WITH AND WITHOUT A HISTORY OF CONCUSSION

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A thesis submitted to the faculty of the University of North Carolina at Chapel Hill in partial fulfillment of the requirements for the degree of Master of Arts in the Department of Exercise and Sport Science (Athletic Training) in the College of Arts & Sciences.

Chapel Hill
2017

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## ABSTRACT

Cally Marie Mackrell: Somatosensory Function in Individuals With and Without a History of Concussion
(Under the direction of J. Troy Blackburn)

Concussion increases the risk of lower extremity injury. This study sought to identify deficits in somatosensory function that may contribute to this heightened risk. Vibratory perception threshold (VPT) and joint position sense (JPS) were compared between individuals with and without a history of concussion. JPS and VPT did not differ between groups (closed kinetic chain (CKC) knee $p = 0.093$; open kinetic chain (OKC) knee $p = 0.255$; open kinetic chain ankle $p = 0.648$). CKC and VPT were correlated at 4 of 5 sites tested ($p = 0.009 – 0.033$). OKC JPS was not correlated with VPT at any of the 5 sites ($p = 0.148 – 0.941$), with the exception of the medial femoral epicondyle and the OKC knee measure ($p = 0.036$). These results do not support our hypothesis. Future research should evaluate the influence of concussion on other characteristics to determine why injury risk is higher following concussion.

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## LIST OF ABBREVIATIONS

| | |
|---|---|
| ACL | Anterior Cruciate Ligament |
| AE | Absolute Error |
| AMEDA | Active Movement Extent Discrimination Assessment |
| CAI | Chronic Ankle Instability |
| CE | Constant Error |
| CKC | Closed Kinetic Chain |
| DAI | Diffuse Axonal Injury |
| FMS | Functional Movement Screen |
| IJPR | Ipsilateral Joint Position Reproduction |
| JPR | Joint Position Reproduction |
| JPS | Joint Position Sense |
| LESS | Landing Error Scoring System |
| MOI | Mechanism of Injury |
| MTBI | Mild Traumatic Brain Injury |
| NWB | Non-Weight Bearing |
| OKC | Open Kinetic Chain |
| RMSE | Root Mean Square Error |
| SAII | Slowly Adapting Type II |
| TTDPM | Threshold to Detection of Passive Motion |
| VE | Variable Error |
| VPT | Vibratory Perception Threshold |
| 3D | Three-Dimensional |

# CHAPTER I: INTRODUCTION

Each year, an estimated 1.6-3.8 million sports-related traumatic brain injuries occur in the United States[1] at an approximated cost of $60 billion.[2] Concussions have been linked to a variety of symptoms[3] that typically resolve in 7-10 days,[4] but may persist longer in 10-15% of cases.[3] Furthermore, those who have suffered a concussion are more likely to suffer a subsequent injury, including another concussion[5-7] and/or lower extremity musculoskeletal injuries.[8 9]

The risk of suffering musculoskeletal injury is 2.2x greater in previously concussed individuals than those who have not suffered a concussion.[8] Risk for suffering an acute lower extremity injury specifically increases two-fold in those with a history of concussion compared to non-concussed counterparts.[9] However, the underlying causes of this heightened injury risk are unclear.

Concussions are considered to be diffuse in nature, potentially affecting the entire brain and causing a variety of symptoms[3] due to a transient disturbance in brain function[10] and axonal shearing.[11] This axonal damage leads to a series of secondary processes that continue to alter neuron function.[11] This widespread altered neuronal function likely explains the wide variety of symptoms experienced in concussion, and may contribute to the increased risk of injury post-concussion. In theory, disruption of the normal function of brain neurons following concussion could lead to alterations in somatosensory function, particularly the sensation of proprioception, or joint position sense. A history of concussion has been shown to affect many major inter-hemispheric, intra-hemispheric, and projection fibre tracts within the brain.[12] If communication between these areas is compromised, somatosensory information may be incorrectly processed and the appropriate decisions for joint positioning cannot be made.

Altered proprioception has been suggested as a contributor to improper joint loading, which potentially contributes to greater injury risk.[13] For example, an individual is more likely to suffer an ACL injury when landing on an extended knee[14] or in extremes of flexion or extension.[15] This suggests that a compromised ability to accurately identify knee joint position might contribute to the joint being placed in a position at which it is at a greater risk of injury. Similarly, Button et al.[16] found that when the foot is in an everted position prior to extreme external rotation of the talocrural joint, the anterior tibiofibular ligament is at a greater risk of failure (high ankle sprain).[17] When the ankle was in a neutral position prior to external rotation of the talocrural joint, sprains of the deltoid ligament were most common.[17] Additionally, subjects with chronic ankle instability (CAI) display greater inversion prior to ground contact during landing,[18] potentially placing the ankle at greater risk of injury. Gait has been shown to be altered in individuals with proprioceptive deficits,[19] and alterations in gait have been identified in individuals with a history of concussion.[20-22] This suggests that individuals with gait alterations following concussion may also have altered proprioceptive senses. Collectively, these data suggest that diminished proprioception following concussion could contribute to the heightened risk of lower extremity injury.

Proprioception has traditionally been measured as joint position sense (JPS) whereby motion capture systems are used to determine an individual's ability to replicate a given joint angle. Unfortunately, these traditional measures of proprioception are not feasible in the clinical setting. However vibratory perception threshold (VPT) provides an indication of somatosensory function that could be a clinical proxy for testing joint position sense. VPT represents the maximum magnitude of vibratory stimulus needed for a subject to experience the sensation, and is thought to follow the same neuronal pathways as proprioception.

A higher VPT has been reported in individuals with knee osteoarthritis who demonstrate improper loading of the knee joint during ambulation, symbolizing a compromised gait pattern.[19] Similarly, proprioceptive deficits have been reported in individuals with knee osteoarthritis.[23] Deficits in gait and dynamic balance have been reported in concussed individuals as well,[20-22] and last anywhere from four to twelve weeks post-injury.[20 22] VPT has also been found to be higher in a cohort of runners with a history of overuse injury compared those without a history of overuse injury.[24] These same runners also demonstrated deficits in JPS,[24] further supporting the potential link between the two measures. If demonstrated to correlate with JPS and discriminate individuals with and without a history of concussion, VPT could be used to assess somatosensory function in the clinical setting and aid in guiding rehabilitation decisions and efforts to decrease the risk of subsequent lower extremity musculoskeletal injury.

In summary, somatosensory function is hypothesized to be compromised following concussion. JPS and VPT are two manners by which somatosensory function can be tested. If concussed subjects display deficits in somatosensory function, a more targeted rehabilitation approach can be taken to minimize injury risk following concussion. Therefore, the purposes of this study were to determine if joint position sense/proprioception and/or vibratory perception threshold (VPT) differ between individuals with and without a history of concussion.

The Specific Aims of this study included the following:

1. To determine the effect of a history of concussion on joint position sense (JPS) at the ankle (OKC) and the knee (OKC and CKC). We hypothesized that subjects with a history of concussion would demonstrate greater JPS absolute error compared to those without a history of concussion.

2. To determine the effect of a history of concussion on vibratory perception threshold, and to determine the relationship between JPS and VPT. We hypothesized that subjects with a history of concussion would demonstrate greater VPT compared to those without a history of concussion.

3. To evaluate the relationship between JPS and VPT. We hypothesized that JPS and VPT will be positively correlated.

# CHAPTER II: REVIEW OF LITERATURE

## Concussion

Concussions are diffuse traumatic brain injuries,[11 12 25-27] and an estimated 1.6 to 3.8 million sports-related traumatic brain injuries occur each year in the United States.[1] Traumatic brain injuries have been estimated to cost over $60 billion per year.[2] Many potential risk factors and causes of concussion have been mentioned in the literature, but the mechanisms of injury (MOI) differ between sports.[28] Player-to-player contact is a common MOI in football, soccer, basketball, lacrosse, and cheerleading; player-to-surface contact is a common MOI in volleyball, wrestling, gymnastics, swimming and diving, and track and field; and player-to-equipment contact is a common MOI in field hockey, lacrosse, soccer, volleyball, baseball, softball, gymnastics, track and field, and swimming and diving.[28] Additionally, Lagolis et. al[1] found that males are twice as likely to experience a concussion.

Traumatic brain injuries have been linked to a variety of symptoms including headache, sensation of "pressure in head," neck pain, nausea or vomiting, dizziness, blurred vision, balance problems, sensitivity to light (photophobia), sensitivity to noise (phonophobia), feeling "slowed down", "in a fog", or "not right", difficulty concentrating, difficulty remembering, fatigue or low energy, confusion, drowsiness, trouble falling asleep, more emotional, irritability, sadness, and nervousness or anxiety.[3]

For most individuals, symptoms resolve in 7-10 days,[4] but in may persist for longer periods in some cases (10%-15%).[3]

Concussions are diffuse in nature, as their external mechanism produces acceleration and deceleration of the brain within the skull, along with rotational and linear forces,[10] causing axonal shearing.[11] The diffuse axonal injury (DAI) seen in concussions is a critical mechanism in understanding the pathophysiological processes that underlay traumatic brain injuries.[25] After the initial moment of injury, the structural and functional changes in the axons initiate a cascade of secondary processes that continue to alter neurons and may persist for months post-injury.[29] Since concussions are diffuse in nature, it is difficult to determine where their effects may manifest.

**Increased Risk of Lower Extremity Musculoskeletal Injury Post-Concussion**

While post-concussive symptoms are seen in only 10%-15% of all cases,[3] concussion may also incur additional repercussions. Lynall et. al.[9] demonstrated that individuals with a history of concussion were nearly 2x more likely to suffer an acute lower extremity musculoskeletal injury compared to those without a history of concussion. Similarly, Nordstrom et al.[8] reported that elite male soccer players who suffered a concussion were at a 2.2x greater risk of suffering a subsequent musculoskeletal injury after returning to play. While muscle injuries were most common in the follow-up period, risk of all types of injuries was increased.[8]

The underlying causes of the heightened risk of musculoskeletal injury following concussion are unclear. Nordstrom et al.[8] reported that while athletes who suffered concussion displayed a higher injury rate in the subsequent year compared to those without a history of concussion, they also displayed a greater injury rate in the year preceding the concussion. This suggests that pre-existing factors may also influence subsequent injury risk.

The increased risk of acute lower extremity musculoskeletal injury following concussion could be linked to a decrease in joint position sense/proprioception in those who have suffered a concussion. It has been suggested that with impaired proprioception, there is improper loading of the joint which potentially contributes to greater injury risk.[13] Since proprioception stems from the integration of somatosensory information in the cerebral cortex, a head injury could potentially alter how information is processed, causing individuals to have improper joint positioning and increasing their potential risk for injury. The position of a limb in space is made aware of via visual and somatosensory information that is converged in neurons in the premotor cortex.[30] The brain is then able to use this information about the joint's position to make decisions about how to guide its motions.[30] Since vision appears to be a key factor in proprioception, deficits in vision following concussion may negatively affect the brain's ability to process somatosensory information and protect the body from injury. While vision impairment may alter processing of somatosensory information, concussions are injuries to the brain and therefore may affect the way the brain itself processes somatosensory information, thus altering proprioception.

Proprioception acuity has been linked with knee pain and function.[13] Of all subsequent injuries following concussion, Nordstrom et al.[8] noted that 13% of the injuries to the concussed cohort occurred in the knee with another 11% encompassing knee/leg/ankle/foot contusions, and 11% occurred in the ankle with 2% occurring in the leg/ankle/foot. Several studies have discussed the potentially threatening positions of the knee that may lead to ACL injury. The ACL is more likely to be injured when the participant lands on an extended knee,[14] or in extremes of flexion or extension.[15] Another vulnerable position of the knee that potentially leads to ACL injury is extension accompanied by an increase in anterior tibial translation, internal rotation of the tibia, and valgus rotation.[14] Proprioceptive training has been proposed as a potential training method to attempt to decrease the risk of injury. Caraffa et al.[31] found that in a group of proprioceptively trained soccer players, the frequency of injury decreased 7-fold when compared to a control group.

In addition to vulnerable positions of the knee, many studies have shown the potentially vulnerable positions of the ankle. When the ankle joint is externally rotated, either a high or medial ankle sprain can occur, depending on the position of the foot.[16 17] Button et al.[16] found that when the ankle is in a neutral position and externally rotated, the risk of a sprain of the deltoid ligament increases (medial ankle sprain). When the ankle is in an everted position and externally rotated, the risk of a high ankle sprain of the anterior tibiofibular ligament increases.[16] Another vulnerable position of the ankle is seen in subjects with chronic ankle instability (CAI) who display greater inversion during side jumping, greater inversion and eversion during vertical drops, and lesser plantarflexion during touchdown compared to healthy individuals.[18]

**Somatosensory Function**

Somatosensory functioning involves the input of a variety of cell types, primarily proprioceptors, mechanoreceptors and Merkel cells. Merkel cells are likely an irreplaceable aspect of the somatosensory system, as they are responsible for light-touch response.[32] Proprioceptors are closely tied to motor function and receive their excitation at their peripheral sensory endings.[33] Articular receptors are proprioceptors specifically located within joints.[33] These receptors are most active when the joint is at an extreme of its range of motion, but are fairly inactive in the middle range, making them unlikely to provide trustworthy information during natural movements.[33 34] Mechanoreceptors, which are specialized nerve endings that provide neural input to the central nervous system[35], also provide input into the somatosensory system. These mechanoreceptors provide information that aids in automatic control of movement, balance and postural control, as well as joint stability.[35]

Mechanoreceptors are afferent neurons and are present in the skin, in muscle, and in joints.[34] The cutaneous receptors have been found to only be sensitive in extreme ranges of motion, making them unlikely contributors to proprioceptive information when a joint is in its mid range.[34] Slowly adapting Type 2 (SAII) afferent neurons are the best cutaneous receptor to help determine joint position.[34] Muscle receptors are another type of afferent neuron that provide unidirectional information of joint movement, however it has been found that large numbers of excited neurons are needed to produce proprioceptive sensations.[34] Joint mechanoreceptors are a third type of afferent neuron that contribute to proprioception.[34] These receptors are classified in two main groups: group 2, large-diameter and rapidly conducting, and groups 3 and 4, small-diameter thinly or unmyelinated.[34] Group 2 consists of Ruffini afferents, which are more sensitive to the flexion side of joints, contributing little information unless a joint is in the extreme of its range of motion.[34] Paciniform afferents are another type of Group 2 neuron that is compression sensitive and provides most proprioceptive information when a joint is rotated to the limit of its range of motion.[34] Afferent neurons in Groups 3 and 4 appear to be more sensitive to pain and typically lack any directional specificity.[34] Several studies have shown that animals with unstable joints learn to adapt their movement strategies, but dogs that are lacking these signals eventually injure their joints.[36,37] These data indicate that accurate sense of joint position requires a combination of inputs from mechanoreceptors in the skin, muscles, and joints.[34]

Somatosensory functioning has not been heavily studied in concussed subjects, but research on the nervous system suggests that concussive injuries may have long-term implications for sensory function and brain health.[38] Lynall et al.[9] demonstrated that concussion leads to a higher risk of lower extremity injury. In order to prevent injury, it is crucial that sensory and perceptual information sustains their integrity so an individual can successfully interact with his/her environment.[38] It is important to look more in depth at how concussion could affect somatosensory function and how those effects potentially influence musculoskeletal injury risk.

**Proprioception**

Proprioceptors are receptors located in skin, muscle, and joints[34,39] that relate information to the brain related to a joint's position in space (i.e. joint position sense). Muscle afferent neurons mediate a large portion of position sense in the body.[34] Senses of proprioception are derived from information in muscles and joints, but they are not experienced in muscles and joints.[39] Position sense via muscle receptors is more accurate when active rather than passive placement is used in the movement of the joint.[40] Muscle spindles are not able to accurately predict the static position of the limb when it has been moved passively,[41] likely because they are dynamic structures. Muscle spindles provide information relative to tissue length, while their counterparts, Golgi tendon organs, provide information as to tissue force.[34] The more superficial a structure or surface, the better humans can determine the localization of the sensation.[39] As receptors get deeper, as they are within joints, it becomes increasingly more difficult to localize sensation, supporting the notion that joint position sense is derived from information in those receptors, and not directly experienced there.[39]

Proprioceptive information is transmitted from sensory receptors in the periphery to the somatosensory cortex and the somatosensory association cortex in the brain.[42] There, it is synthesized and processed, then transmitted to the motor cortex where the brain takes the new information and information from previous experiences to determine the proper movement of the joints and activation of muscles.[42] The motor cortex itself has two separate areas, the premotor cortex and the supplementary motor cortex.[42] The primary job of the premotor cortex is to use external cues from the environment to guide movement.[42] The supplementary motor cortex uses memory to induce coordination and achieve motor goals.[42] If either of these two areas is interrupted or altered as a result of concussion, the brain's ability to control the body is compromised. Parietal areas of the cerebral cortex are another large source of input for the somatosensory system.[42] The information generated by the parietal areas is related to kinesthesia, or the relative position and movement of body segments.[42] A history of concussion has been shown to affect many major inter-hemispheric, intra-hemispheric, and projection fibre tracts within the brain.[12] If these areas are unable to accurately communicate with each other, somatosensory information cannot be processed correctly and the appropriate decisions for joint positioning are compromised.

The potential causes of the increased risk of lower extremity musculoskeletal injury are not well represented in the literature. Several studies have shown deficits in gait following concussion, as well as deficits in dynamic balance.[20-22] These demonstrated deficits could potentially be linked to insufficiencies in somatosensory function, which may lead to alterations in proprioception and positioning of the joints, leading to a greater risk of injury. Pietrosimone et al.[43] suggested that the deficits in neurological and neuromuscular functioning following a concussion might be due to injury of intracortical neurons. It was also hypothesized that altered movement patterns due to one type of injury (concussion or lower extremity) increases the risk of the other.[43] Additionally, those with a history of concussion display slow motor execution on a diadochokinesia task,[44] which could delay their ability to move out of the way of harm, thus increasing their risk for injury.

De Ridder et al.[18] studied subjects with chronic ankle instability (CAI), copers, and a control group and found differences in foot/ankle position during landing tasks and side jumping. In landing and jumping tasks, the ankle's position as it is coming in contact with the ground is important. Since ankle injuries are most common during jumping, landing, and cutting activities,[45] it is appropriate to test the joint in a NWB position, since the injuries commonly occur when landing in a potentially compromising position.

**Evaluating Somatosensory Function: Proprioception**

Measurements of proprioception vary depending on which joint is being tested and what equipment is utilized, as well as the specific research question. When evaluating proprioception and joint position sense, four common measures are typically used including absolute error, constant error, variable error[46 47] and root mean square error.[47 48] Absolute error (AE) disregards the direction of error (overshoot or undershoot)[46] and reflects the total deviation from the starting point.[47] Constant error (CE) is similar to AE in that it is a comparison of error, but unlike AE, CE takes the direction of error into account.[46 47] The standard deviation of constant error, indicating consistency about the mean, is defined as variable error (VE).[46 47] Root mean square error (RMSE) is equal to the square root of the sum of the CE squared and the VE squared.[48] The RMSE gives the researcher an overall measure of how successful the subject was in achieving the target position.[48]

Knee joint position sense is typically evaluated using one of three pieces of equipment: an isokinetic dynamometer, an electrogoniometer, or 2D motion analysis. Both the electrogoniometer and 2D video analysis produce excellent correlation when the subject is tested in a functional and weight-bearing position.[49] The knee is placed in a reference position, and the subject is then asked to reproduce the reference angle, and the accuracy of reproduction is determined by the motion analysis system.[49]

When evaluating ankle joint position sense, three common methods are seen throughout the literature: threshold to detection of passive motion (TTDPM), joint position reproduction (JPR), and active movement extent discrimination assessment (AMEDA).[50] The TTDPM involves the subject seated or laying down with the joint of interest in a machine controlled by the investigator.[51] The investigator controls the speed of joint movement while the subject presses a button once he/she senses movement and direction of the joint.[51] The subject is then instructed to state the perceived direction of movement, and if incorrect, the trial is discarded.[51] JPR testing involves the ipsilateral and/or contralateral limbs and can be tested passively or actively.[52] Ipsilateral joint position reproduction (IJPR) involves a subject being moved to a target position actively or passively, returning to the initial position, and attempting to reproduce target angle in the same manner in which it was shown.[50 52] One method of contralateral joint position reproduction involves the same concept as IJPR, however the contralateral limb attempts to reproduce the target angle.[50 52] In the third JPR method, one side positions the limb in the target position and holds it there while the contralateral side attempts to match the held position.[50 52] Finally, the AMEDA test is conducted using active motion in which the subject's limb is placed in the AMEDA apparatus and they are introduced to five joint displacements. [50 53] The subject then completes 50 trials where each position is presented ten times in a random order.[50 53] When the joint is in position, subject is required to state which of the five displacements he/she believes the joint is in, and the ability to correctly judge the position is assessed.[50 53]

The validity of the ankle proprioception measures mentioned above varies greatly. JPR tests in general have low testing validity because the proprioceptive information available during target positioning differs from that available during target reproduction.[54] The TTDPM testing only records "correct" trials and discards those in which the subject does not successfully reproduce the position.[55] Few studies have reported the number of incorrect trials or the percentage of trails guessed correctly,[50] making it hard to determine validity of the testing procedure. Finally, the number of trials used can provide issues in regards to validity of the testing protocol. The TTDPM and JPR involves typically use 3-5 trials,[55 56] whereas the AMEDA testing protocol calls for 50 trials.[50] The difference in number of trials used can have an effect on the validity of the test.

**Evaluating Somatosensory Function: Vibratory Perception Threshold**

Perception of vibratory sensation travels along neurologic pathways similar to those of proprioception. "Vibratory sensation may be an important component in providing tactile feedback to the central nervous system during ambulation, and alterations in vibratory acuity could affect the kinetics of the lower extremity."[19] Testing of vibratory perception threshold (VPT) typically uses a biothesiometer. In order to assess VPT, five test sites are commonly used: the 1st metatarsophalangeal joint, medial malleolus, lateral malleolus, medial femoral condyle, and lateral femoral condyle.[57] Uniform pressure is essential during testing and can be achieved by using the weight of the biothesiometer itself as the sole source of pressure.[57] Once the patient is in place and the biothesiometer is prepared, the voltage begins at 0 and is gradually increased at a rate of 1 Volt per second.[57] The patient then verbally or manually indicates when he/she first experiences the sensation of vibration.[57] The voltage of the biothesiometer at the initial sensation of vibration is called the vibratory perception threshold.[57] It is normal for an individual's VPT to increase as testing site moves from distal to proximal.[57] Measurement of VPT using a biothesiometer demonstrates high reliability.[57] In multiple studies, patients with osteoarthritis displayed greater VPT at all testing sites compared to healthy control subjects.[57] [58] As these individuals typically display proprioceptive deficit,[19] VPT appears to be a surrogate measure of proprioception.

Vibratory perception threshold is also altered in osteoarthritic subjects who demonstrate higher loading of the knee joint during gait.[19] These specific findings were most relevant to changes in VPT at the first metatarsophalangeal joint, hypothesized to be the result of the first metatarsal being the best anatomical site to appreciate the sensation of the foot making contact with the ground during gait.[19] Similar alterations in gait were seen in subjects who had suffered a concussion.[20-22] Therefore, changes in sensory perception following concussion may contribute to compromised gait patterns and greater injury risk.

In summary, it has been hypothesized that because concussions are diffuse in nature and their effects are widespread, the processing of somatosensory information in the brain may be compromised post-concussion. This compromised somatosensory function may contribute to the heightened risk of musculoskeletal injury following concussion. To our knowledge, there have not been any studies looking at the link between vibratory perception threshold and proprioception, making this the first to attempt to make a connection. Although proprioception is a difficult sense to measure in the clinical setting, VPT may be a more clinically applicable means by which to measure potential proprioception and somatosensory function post-concussion.

## CHAPTER III: METHODS

### Experimental Design

A cross-sectional laboratory study design was utilized to compare lower extremity somatosensory function between individuals with and without a history of concussion. Subjects completed a single testing session during which vibratory perception threshold (VPT) and lower extremity proprioception (knee and ankle joint position sense [JPS]) were assessed bilaterally. The order of assessments was determined via a balanced Latin square.

### Subjects

*A priori* power analysis indicated that a sample of 36 subjects would provide power of 0.80 to identify differences in somatosensory function between individuals with and without a history of concussion ($\alpha = 0.05$). However, these calculations were based on two studies that were not directly applicable to the present study. Shakoor et al.[57] evaluated VPT in 27 subjects with osteoarthritis (OA) and 14 healthy subjects, and reported greater VPT in OA subjects [57]. Cossich et al.[59] reported greater JPS errors in the ACL-deficient limbs of 20 subjects with unilateral ACL injuries relative to the healthy contralateral limb [59]. Since the two studies above were not directly representative of the population of interest in the present study, a sample size of 50 (25 in each group) was recruited to ensure adequate statistical power. During the time period of data collection, it was difficult to recruit 50 qualified subjects, so a sample size of 20 was obtained. During the recruiting process, potential subjects were contacted without reply. Twenty-two potential subjects were identified and contacted for the concussed cohort, but only 12 responded and were subsequently tested, with 10 used in the current study. Primary investigator also attended various club practices and classes to recruit without success. Though the sample size does not display adequate power, it provides preliminary data regarding the influence of concussion on somatosensory function.

Twenty subjects (10 male and 10 female) were recruited for this study based on their concussion history. Subject demographics are provided in Table 1. A total of 10 subjects (Concussed Cohort) had suffered a clinician-diagnosed concussion within the year prior to participation, while the remaining 10 subjects (Control Cohort) had no history of concussion. Additionally, subjects in the Control Cohort were matched to subjects in the Concussed Cohort for sport participation/activity level and sex. The Tegner activity scale was used to assess activity level, and all subjects were required to score a minimum of five (i.e. physically active or participating in recreational sports at least two days a week). Additionally, all subjects were required to have no history of acute lower extremity musculoskeletal injury within the 6 months prior to participation, chronic musculoskeletal conditions (e.g. patellofemoral pain, chronic ankle instability, etc.), or lower extremity surgery. Subjects in the Concussed Cohort were included if their most recent concussion occurred within 1 year (365 days) of participation, they were asymptomatic, they had been cleared for return to full participation in physical activity, and they had fully returned to play for at least two weeks. All participants signed an IRB-approved informed consent form prior to being enrolled in the study.

**Procedures**

Upon arrival to the laboratory, subjects warmed up for 5 minutes on a cycle ergometer at a self-selected pace and resistance. Vibratory perception threshold (VPT) was evaluated using a biothesiometer that consisted of a vibrating tip that moved at a constant frequency of 120 Hz and a manual dial that was used to adjust vibration intensity. Subjects were positioned side-lying on a padded table, and the biothesiometer was applied uniformly to four bony processes of the lower extremity (medial and lateral epicondyles, and the medial and lateral malleoli) with the weight of the device itself being the only source of pressure. A fifth bony process, the base of the first metatarsal, was assessed with the subject seated with the feet flat on the floor. Prior to testing, the subject was given a demonstration of the effect of the biothesiometer on the hand to familiarize him/her with the vibratory sensation. With the biothesiometer placed at the testing site and the voltage/intensity set to 0, the intensity was increased at a rate of 1 V/s, and the subject was instructed to verbally indicate when he/she first sensed the vibration. The corresponding voltage was then recorded as the VPT. Three trials were conducted at each testing site and averaged for statistical analysis.

Proprioception of the ankle and knee joints was evaluated by measuring active JPS. Knee JPS was evaluated in both the open and closed kinetic chains, while ankle proprioception was measured in the open kinetic chain only. The joint being tested was placed in a reference position randomly determined by the investigator prior to each trial. The subject was instructed to maintain the reference position for 3-5 seconds, return to the starting position, and then attempt to actively replicate the reference angle. The absolute error between the reference angle and the angle produced was averaged across five trials.

Kinematics were sampled during JPS testing using the Motion Monitor motion capture system. Subjects were instrumented with 6 electromagnetic motion tracking sensors placed bilaterally on the lateral aspect of the mid-thigh, the anterior aspect of the shank, and the dorsal surface of the foot. The medial and lateral femoral condyles, medial and lateral malleoli, and $2^{nd}$ phalanx were digitized to create a segment-linkage model of the lower extremities.

For the closed kinetic chain assessment of knee JPS, subjects were positioned supine on a sliding platform reclined 30° relative to the horizontal with a wedge placed under the testing limb placing the ankle in a slightly plantarflexed position to minimize the potential for gastrocnemius tightness to restrict knee motion. Starting in full knee extension, the subject actively flexed the knee while the investigator viewed the joint angle in real time and verbally indicated when the subject reached the target angle which was randomly determined (20°, 25°, or 30°) prior to each trial. The subject was then instructed to press an electronic trigger to provide a time stamp for the kinematic data, and to then return to the starting position (i.e. full knee extension) for 5 seconds. During this interval, the subject donned a blindfold and headphones providing white noise to eliminate visual and auditory cues. The subject then attempted to flex the knee and recreate the target angle, and pressed the electronic trigger when he/she perceived the target angle was reached. This method was repeated five times per limb.

These same procedures were repeated for the ankle and knee with the subject in a non-weight bearing position. The subject was supine on a table with the legs not touching the ground. For the open kinetic knee measure, normal resting position for the subject was used as the starting position for testing, with the knee in ~90º of flexion and the ankle loosely plantarflexed off the end of the table. For the open kinetic ankle measure, the same supine position was used, though the patient had a foam roller placed under the back of the knees to eliminate gastrocnemius tightness from preventing full ankle range of motion, with only the distal half of the shank off the end of the table and the ankles loosely plantarflexed. Prior to beginning a trial for the ankle assessment, the subject was instructed to fully dorsiflex the ankle. The subject returned to this position prior to attempting to recreate the target angle. Target angles for ankle JPS included 15° and 30° of plantarflexion, while knee JPS was assessed at the same angles as for the closed chain assessment (20°, 25°, and 30° of flexion).

**Data Processing**

Kinematic data were sampled at 200 Hz and lowpass filtered at 10 Hz ($4^{th}$ order Butterworth). The average Grood and Suntay joint angle during the 500ms following each time the trigger was pressed was calculated to identify the reference angle and the reproduced angle. The absolute error was calculated as the absolute value of the difference between the reference angle and the reproduced angle.

**Statistical Analyses**

The data were checked for normality using the Shapiro-Wilk test, visual inspection of the histograms, and evaluation of the ratio of skewness and kurtosis statistics to their standard errors. A series of independent samples t-tests were used to compare VPT and ankle and knee JPS of the dominant limb between the Concussed and Control cohorts. Dominant limb data was used as the only mean of comparison due to time restraints during the analysis process. Pearson product-moment correlations were used to evaluate relationships between VPT at each testing site and JPS at the ankle and knee, respectively. Statistical significance was established *a piori* as $\alpha = 0.05$.

## CHAPTER IV: RESULTS

### Joint Position Sense

JPS did not differ between the groups in the closed kinetic chain knee assessment ($t_{18}$ = -1.78; p = 0.093), open kinetic chain knee assessment ($t_{18}$ = 1.18; p = 0.255), or the open kinetic chain ankle assessment ($t_{18}$ = 0.47; p = 0.648). Descriptive statistics for the JPS comparisons are provided in Table 2.

### Vibratory Perception Threshold

VPT did not differ between the groups at the medial femoral epicondyle ($t_{18}$ = -0.19; p = 0.849), lateral femoral epicondyle ($t_{18}$ = 0.66; p = 0.521), medial malleolus ($t_{18}$ = -1.75; p = 0.097), lateral malleolus ($t_{18}$ = -1.03; p = 0.316), or the base of the first metatarsal ($t_{18}$ = -0.89; p = 0.387). Descriptive statistics for the VPT comparisons are provided in Table 3.

### Correlations

Correlations between JPS and VPT are presented in Table 4. There was a significant correlation observed between closed kinetic chain knee JPS and VPT at four of the five sites tested (r = 0.433 – 0.565). Poorer knee JPS during the closed kinetic chain assessment, indicated by a higher absolute error, was associated with poorer VPT, indicated by higher VPT values. This association was significant at all testing sites with the exception of the lateral femoral epicondyle, which approached statistical significance (p = 0.057). These correlations suggest that JPS and VPT reflect similar phenomena. A significant correlation was also noted between VPT at the medial femoral epicondyle and JPS at the knee in the open kinetic chain ($p = 0.036$), but not at the other four testing sites. No significant relationships were found between VPT and the open kinetic chain ankle JPS measurement.

# CHAPTER V: DISCUSSION

Sustaining a concussion increases the risk of suffering an acute lower extremity injury upon return to sport.[8 9 60] The ability to identify the underlying causes of this increased risk may allow for the implementation of targeted rehabilitation for individuals returning to sport from concussion. Our findings did not support our hypothesis that joint position sense (JPS) and vibratory perception threshold (VPT) are compromised up to one year following concussion. We identified significant correlations between the closed kinetic chain knee JPS assessment and 4 of the 5 VPT testing sites, suggesting that these measures reflect similar phenomena. However, there were no significant correlations between VPT at any of the testing sites and the two open kinetic chain JPS measures. During the open kinetic chain measure, while the knee was the joint of interest, proprioceptive feedback is received from the ankle and the hip and can also be used to indicate the position of the knee. While assessing the open kinetic chain joint position sense, proprioceptive feedback is only received from the knee, limiting the amount of information available to use for decision-making.

While our study was the first to evaluate lower extremity JPS following concussion, our findings are in agreement with those of Hides et al.[61] who examined cervical JPS in rugby players who had sustained a concussion. These authors did not find any differences in cervical JPS between the baseline assessment and the assessment taken 3-5 days post-injury.[61]

To our knowledge, the present study is also the first to evaluate VPT following concussion. VPT was not different between groups, but was correlated with the closed kinetic chain JPS measure. With the knowledge that these two measures are correlated, VPT may be useful as a proxy to assess somatosensory function in the clinical setting. In the future, if proprioception is found to be a relevant risk factor for increased injury risk, testing via laboratory methods involving 3D motion capture equipment is not realistic in the clinical setting due to the cost of the system and lack of measurement expertise. Since VPT was correlated with a JPS measure assessed using this laboratory method, the biothesiometer used to assess VPT is a smaller, less expensive piece of equipment that would make the measure clinically applicable and a more realistic assessment.

Assessing somatosensory function via VPT may be useful for tracking recovery and efficacy of rehabilitation, as several injuries incur deficits in somatosensory function. For example, several studies have shown that proprioceptive abilities decrease following ACL injury.[62-64] Whether the subject is ACL-deficient[63] or underwent ACL reconstruction,[62 64] the ACL limb demonstrates greater error in joint position sense. Proprioceptive deficits have also been reported following ACL injury during both joint movement and joint position testing.[65] With the knowledge that this proprioceptive deficit exists following ACL injury, further research can explore VPT following ACL injury to determine if correlation between VPT and JPS applies when JPS is deficient. This knowledge might also be useful in the clinical setting because VPT may be able to serve as a surrogate measure for JPS, since JPS testing is not feasible in the clinical setting. If we are able to identify deficits in JPS in the clinic, we can target rehabilitation plans to improve joint position sense. We can also use VPT throughout the rehabilitation process to determine if the treatment is effective in improving VPT and subsequently improving JPS.

Several studies have reported an increased lower extremity risk following concussion.[8 9 60] However, compromised somatosensory function does not appear to be a contributor to this risk, as our data indicate it is similar in those with and without a history of concussion. Further studies should explore other potential causes for this increased risk of lower extremity injury. Postural stability and reaction times are two factors that may be explored further in this population. Gonell et al.[66] demonstrated that the Y-Balance test successfully identified soccer players who were at a greater risk of injury, and Dingenen et al.[67] used double leg to single leg transition task to identify female athletes with decreased postural stability that were at a greater risk of suffering a noncontact lower extremity injury. Testing postural stability may provide further insight into potential causes for increased injury risk in the concussed population. Visuomotor reaction time was identified as a modifiable risk factor for suffering an injury in football players.[68] Testing reaction time upon return to play after concussion may provide insight into injury risk, but may also identify a modifiable issue.

The Functional Movement Screen (FMS) and Landing Error Scoring System (LESS) have been proposed as potential screening tools for lower extremity injury risk. Research suggests that the FMS test may be effective for predicting lower extremity injury risk[69], as is the LESS test.[70] All measures mentioned have been shown to identify those at an increased risk for suffering an injury in a specific population. Testing these measures in a concussed population may identify potential causes for the increased injury risk following concussive injury. In addition, testing all of the above measures in a fatigued state is also worth investigating, as fatigue is thought to be an additional risk factor for lower extremity injury.[71-73] All aforementioned tests examine injury risk in functional positions. While JPS is an accurate way to evaluate the somatosensory system, the injury risk increase that is seen after concussion may be the result of functional deficits resulting from disruption of the neuromuscular system as a whole. Research has shown that neuromuscular control is a modifiable risk factor for general knee injury[74] and ACL injury.[74 75] Knowing that neuromuscular training decreases risk of knee injury and specifically ACL injury, future studies should look to the system as a whole in previously concussed individuals.

The present study looked only at the function of the somatosensory system and the ability of an individual to process sensory information. Though no deficits in somatosensory function were identified, future studies should investigate the influence of concussion on motor function. Miller et al.[76] found that after mTBI, while there are occasionally changes in cortical excitability of the motor cortex, there are significant changes in intra-cortical inhibition of the motor cortex for months to years post-injury. Powers et al.[77] also found a decrease in intra-cortical facilitation of the motor cortex following concussion. With greater impulses required in subjects with a concussive history to generate the same response as in healthy subjects, further studies should investigate if a similar hypoexcitability exists in the lower extremity.

The present study has several limitations. The sample size was small which decreases the power of the findings. Caution should be taken when generalizing the findings put forth by this study. Correlations were also only seen at 4 of the 5 sites and only during the CKC assessment. Because the findings were not widespread, it cannot be said that VPT can be used as a proxy for all methods of JPS testing. At the time of testing, 8 of the 10 subjects in the concussed cohort had suffered their injury in the 6 months prior to their testing session. While increased injury risk has been shown up to 365 days post injury[8 9], decreasing the amount of time since injury may yield different results. Brooks et al.[60] found that injury risk was increased during the 90-day period following concussion. Limiting the time since injury to 90 days may show more differences between groups than a larger post-injury time frame.

In conclusion, joint position sense and vibratory perception threshold do not differ between individuals with and without a history of concussion. Higher vibratory perception threshold is correlated with higher absolute error in closed kinetic chain testing of the knee joint and may be able to be used as a clinical proxy for JPS in the closed kinetic chain. We are still unsure as to why the risk for suffering a lower extremity injury increases after concussion, but it does not appear to be due to poor joint position sense.

**Table 1.** Mean ± SD demographic information for subjects.

| | Age (years) | Height (cm) | Weight (kg) | Months Post-Injury |
|---|---|---|---|---|
| Healthy (n=10) | 21.2 ±1.55 | 175.32 ±12.62 | 77.02 ±13.01 | N/A |
| Concussed (n=10) | 20.9 ±2.18 | 166.24 ±10.90 | 68.81 ±9.27 | 4.4 ±3.99 |

**Table 2.** Mean absolute error (degrees) in joint position sense between individuals in the concussed cohort and those in the healthy cohort.

| | Closed Kinetic Chain | Open Kinetic Chain Knee | Open Kinetic Chain Ankle |
|---|---|---|---|
| Healthy | 3.27 ±1.57 | 2.42 ±0.93 | 4.35 ±1.94 |
| Concussed | 2.28 ±0.78 | 3.01 ±1.29 | 4.84 ±2.65 |
| *P* | 0.093 | 0.255 | 0.648 |

**Table 3.** Mean vibratory perception threshold (Volts) at each site between individuals in the concussed cohort and those in the healthy cohort.

| | Medial Femoral Epicondyle | Lateral Femoral Epicondyle | Medial Malleolus | Lateral Malleolus | Base of 1st Metatarsal |
|---|---|---|---|---|---|
| Healthy | 14.81 ±5.78 | 13.30 ±6.18 | 11.73 ±4.65 | 10.77 ±3.90 | 6.50 ±3.29 |
| Concussed | 14.37 ±4.45 | 14.90 ±4.62 | 9.03 ±1.47 | 9.37 ±1.77 | 5.47 ±1.67 |
| *P* | 0.849 | 0.521 | 0.097 | 0.316 | 0.387 |

**Table 4.** Correlation of vibratory perception threshold and joint position sense at five sites of the lower extremity in individuals with and without a history of concussion.

| | Medial Femoral Epicondyle | Lateral Femoral Epicondyle | Medial Malleolus | Lateral Malleolus | Base of 1st Metatarsal |
|---|---|---|---|---|---|
| **CKC** | | | | | |
| Pearson r | 0.470 | 0.433 | 0.553 | 0.478 | 0.565 |
| *P* | **0.033** | 0.057 | **0.011** | **0.033** | **0.009** |
| **OKC Knee** | | | | | |
| Pearson r | 0.470 | 0.335 | -0.054 | 0.018 | 0.114 |
| *P* | **0.036** | 0.148 | 0.821 | 0.941 | 0.633 |
| **OKC Ankle** | | | | | |
| Pearson r | -0.052 | -0.150 | -0.200 | -0.269 | -0.148 |
| *P* | 0.829 | 0.529 | 0.397 | 0.252 | 0.533 |

*CKC = closed kinetic chain*
*OKC = open kinetic chain*

APPENDIX 1: TEGNER ACTIVITY SCALE

| | |
|---|---|
| **Level 10** | **Competitive sports- soccer, football, rugby (national elite)** |
| **Level 9** | **Competitive sports- soccer, football, rugby (lower divisions), ice hockey, wrestling, gymnastics, basketball** |
| **Level 8** | **Competitive sports- racquetball or bandy, squash or badminton, track and field athletics (jumping, etc.), down-hill skiing** |
| **Level 7** | **Competitive sports- tennis, running, motorcars speedway, handball**<br><br>**Recreational sports- soccer, football, rugby, bandy, ice hockey, basketball, squash, racquetball, running** |
| **Level 6** | **Recreational sports- tennis and badminton, handball, racquetball, down-hill skiing, jogging at least 5 times per week** |
| **Level 5** | **Work- heavy labor (construction, etc.)**<br><br>**Competitive sports- cycling, cross-country skiing,**<br><br>**Recreational sports- jogging on uneven ground at least twice weekly** |
| **Level 4** | **Work- moderately heavy labor (e.g. truck driving, etc.)** |
| **Level 3** | **Work- light labor (nursing, etc.)** |
| **Level 2** | **Work- light labor**<br><br>**Walking on uneven ground possible, but impossible to back pack or hike** |
| **Level 1** | **Work- sedentary (secretarial, etc.)** |
| **Level 0** | **Sick leave or disability pension because of knee problems** |

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